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THE BIOMEDICAL EFFECTS OF THE HYPERBARIC ENVIRONMENT.(U)

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)
Hyperbaric bradycardia was observed in 3 of 4 subjects at rest and during exercise at 5.5 Ata while breathing normoxic helium with a gas density equivalent to air at 1 Ata. The bradycardia was evident within 1 hour after reaching the simulated depth but was not present after 24 hours of exposure. Cardiac output at rest and during exercise after 24-48 hours at 5.5Ata was not significantly different from the control values at 1 Ata; the bradycardia

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was not present at these times.

Airway pressures during the first 100 msec of an occluded breath were greater at 5.5 Ata than at 1 Ata. The data indicate an increased respiratory center motor output at increased hydrostatic pressure. The response may be due either to the increased pressure or to inhalation of a gas with an increased density just prior to the occlusion.

Hyperbaric bradycardia was not observed in five subjects after rapid compression to 5.5 Ata at which depth the subjects were breathing normoxic helium.

The Biomedical Effects of the Hyperbaric Environment
ONR Contract N000-14-67-A-0251. Aug. 1, 1974-July 31, 1975
H.A. Saltzman, Prinicpal Investigator
J. Salzano and E. Camporesi, Co-Investigators

The goal of these experiments was to learn more about the repeatedly observed alterations in respiration and heart rate at simulated depths.

Four healthy males with some diving experience were used as subjects. Control measurements were made at 1 Ata while subjects breathed air. All control measurements were repeated on the subjects at a pressure of 5.48 Ata in a gaseous environment of 3.6% O_2 and helium. Under these conditions the PO_2 and gas density were equivalent to inspired air at 1 Ata.

The time course for the development of hyperbaric bradycardia was studied during the first day of the dive. Heart rates were significantly less than surface controls at rest and during exercise during the first six hours of the hyperbaric exposure in 3 of the 4 subjects. Heart rates at rest and during exercise, however, were not significantly different from controls on the second and third day of exposure. Cardiac output measured by the dye dilution technique was not different from surface controls on the second and third day of exposure. Cardiac outputs were not measured during the first day of the dive. The data indicate an immediate development of bradycardia as a result of the hyperbaric exposure in some subjects, a loss of the phenomenon when exposure is continued and no change, relative to 1 Ata, in cardiac output when the heart rate returned to values observed at 1 Ata. Since we did not anticipate that heart rates would return to control levels on the days that cardiac output was measured we do not know if a heart rate dependent decrease in blood flow and blood pressure occured on the first day of exposure at 5.48 Ata.

To assess the sensitivity of the respiratory centers at depth, the ventilatory response to CO_2 (Modified Read Technique) was measured in subjects breathing a CO_2, O_2 gas mixture in either N_2 or He at 1 Ata and while breathing CO_2, O_2 in helium at 5.5 Ata. Airway pressure, 0.1 sec after the onset of an occluded inspiration, ($P0.1$ sec, modified Milic-Emili Technique), was determined. A greater ventilatory response occurred with the CO_2-He mixtures than with the CO_2-N_2 mixtures at 1 Ata ($p < .05$) which was attributed to the lesser density of the CO_2-He mixtures. Sensitivity to CO_2-He at 5.5 Ata was the same as to CO_2-N_2 at 1 Ata in which case the inspired gas densities were the same. The occulsion pressure was greater at 5.5 Ata when compared to the value at 1 Ata obtained with helium as the inert gas. These data indicate an increased neural output to inspiratory muscles at increased barometric pressure, if the mechanical properties of the respiratory system remained unchanged. Although the

occulsion pressure at 100 msec is a static measurement and therefore not affected by the inspired gas density the increased effort at 5.5 Ata may reflect a pre-programing of the respiratory center to a given gas density. This cannot be ruled out in these experiments because the control measurements at 1 Ata were with helium as the inert gas and the measurements at 5.5 Ata were also with helium. Hence the gas density was not the same in the two conditions.

Arterial blood gases were measured at rest and during graded exercise in subjects breathing air at 1 Ata (Control) and while breathing normoxic-helium at 5.48 Ata. Resting $PaCO_2$ measured every two hours in each subject during the first 8 hours of exposure was not statistically different from control. However, after 24 or 48 hours of exposure resting $PaCO_2$ was 2.5 Torr greater than control ($P < .05$, based on paired values). $PaCO_2$ during exercise at 50W, 100W, and 130W, was greater than control after 24-48 hours at pressure; 2.3, 2.0, and 1.7 Torr, respectively. The increases are statistically significant ($p < .05$) for 50W and 100W. The results indicate that more than 8 hours of exposure to this hyperbaric environment are required for a significant increase in resting $PaCO_2$. Since gas density and oxygen pressure at 5.48 Ata were the same as at 1 Ata the causative agents appear to be either hyperbaric helium or hydrostatic pressure. The mechanism by which either agent provokes the phenomenon is not apparent.

The Biomedical Effects of the Hyperbaric Environment
ONR Contract N00014-75-C-0223. Aug. 1, 1975-July 31, 1976
J. Salzano, Principal Investigator
H. Saltzman, E. Camporesi-Co.-Investigators

To further pursue questions unanswered in the initial year of the contract a renewal was granted for Aug. 1, 1975-July 31, 1976.

During this year occlusion pressures were measured in a group of subjects during rebreathing either CO_2-He gas mixtures or CO_2 gas mixtures at 1 Ata. The data showed that, as in the previous study, the ventilatory response to a given CO_2 pressure was greater with helium than with nitrogen as the background gas. More importantly, the occulsion pressure for a given CO_2 pressure was greater with the more dense (CO_2-N_2) gas mixtures. Again, since the measurement is made under static conditions it cannot reflect a decreased response to an increased respiratory load, i.e. greater density with CO_2-N_2, but most probably reflects a pattern set in the respiratory center as a result of breathing a more dense gas just prior to the applied airway occlusion. Therefore, the data from the previous year which indicated an increased respiratory neural output for a given CO_2 at 5.5 Ata most probably does not entirely represent a hydrostatic pressure effect

on respiratory center motor output.

Also during this year it was planned to perform a saturation dive to 5.5 Ata with gas conditions the same as used in the previous dive. Cardiac output was to be measured in the divers immediately upon reaching the bottom. This would be, according to the previous data, at the time the subjects were experiencing hyperbaric bradycardia. Because only 3 of the 4 subjects in the previous dive exhibited bradycardia it was felt that a larger group of subjects would be needed in order to obtain statistically valid data. Since the bradycardia disappeared within 24 hours under the conditions of the dive it was not logistically possible to do the experiment on a large group of subjects in this time span while using the dye dilution technique for measuring cardiac output. The main problems being the necessary venous and arterial catheterizations and their maintenance in such a large group. We therefore decided to use a bounce dive approach with one subject per dive. Heart rate was measured during various exercise levels while the subject breathed air at 1 Ata over a period of days until a reliable baseline was established. The subject would then dive to 5.5 Ata with the prescribed heliox gas supplied through the inspiration line. Heart rate was measured during exercise during the first hour on the bottom. The rationale was to avoid unnecessary catheterization and prolonged decompression. If a subject developed hyperbaric bradycardia he would come back to the laboratory on a different day, be catheterized and cardiac output and heart rate measured at rest and during exercise at 1 Ata then immediately afterward dive to 5.5 Ata and the measurements repeated. Such studies were done in five subjects. For reasons not at all understood none of the subjects exhibited hyperbaric bradycardia under the conditions of this experimental design. It was thought that it might be related to being compressed with air rather than with heliox as in the original experiment, although the inspired gas composition was the same in both dives. We, therefore, studied two of the five subjects at 5.5 Ata after compressing the chamber with heliox. The subjects did not have heart rates different from the surface control values.

At this time the contract year expired and a no-cost one year extension was granted.

Another approach was designed. We selected two subjects who could maintain an exercise load of 50 Watts for a period of one hour while heart rate was continuously measured. The subject worked at this level in the chamber at 1 Ata until a steady state was established, about 8 minutes. The chamber was rapidly pressurized while the subject continued the activity. At 5.5 Ata he was switched to the

desired breathing mixture. Under these conditions one subject experienced a tachycardia while the other had no significant change in heart rate.

At this time it was decided to search for a non-invasive, less time consuming method to measure cardiac output and to use this method in a large group of subjects in a dive designed exactly as the original one. We were not convinced that the non-invasive techniques available were valid in a hyperbaric environment and the experiment was delayed until such validation could be made. Due to other commitments we were not able to do this before the one-year extension time expired.

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